Electrophysiology

(Cardiac muscle : intrinsically activate itself)

Automaticity (Spontanously depolarize & trigger action potential to spread & contract) [intrinsically]

Myocardium → 2 prts : 1 Nodal cells → Set a rythym
- SA node
- AV node
- AV bundle \ bundle branches
- Purkinji fibers

2 Contractile Cells (actin & myosin)
- Sarcoplasmic reticulum etc ✓

in case of SA disfunction : 2nd is AV & 3rd is Perkinji

SA Node → Sinus rythym = 60-80 bpm on its own no sympathetic\parasympathetic effect

Generally send action potential \ sends to all muscle by conduction pathway : to contract

How does it happen? آكل طاله مارح ينفع

What is it ? ← answers are coming ↓

✡ so: How is the Contraction of Cardiac muscle happen?

Stimulus → to → SA node {Conduct it} → to → Av node & bundle → Contraction

What is the Stimulus & how does it Respond → 1 Automaticity + rruythmicity, 2 Excitability

How the conductive system occur? → 3 Conductivity

How → 4 Contractility

} Cardiac propertis

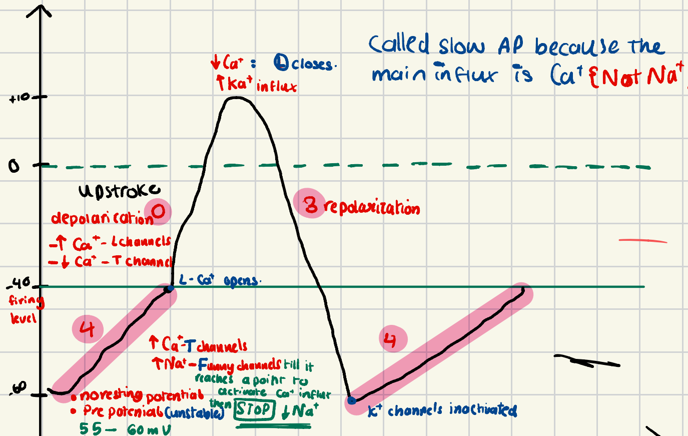
1 Automaticity

define
- Properties of self-excitation :
- Spontanous action potential generation
- Rhythmicity regulate AP generation

Modified non contractive cells :

Pacemakers : highest rythmicity Part.

1. SA node
   - Sinus rhythm
   - Highest [90-110 per min]
2. AV node
   - nodal rhythm [45 - 60 per min]
3. Purkinji
   - idio ventricular rhythm [25-40 per min]

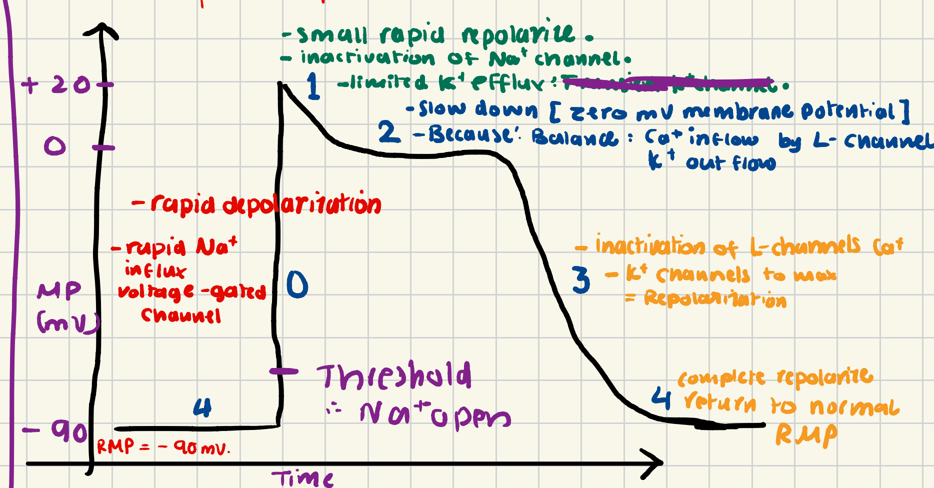


✡ unstable membrane potential
✡ slow upstoke
✡ dur : 200 - 250 ms
✡ no Plateau

2 Excitability

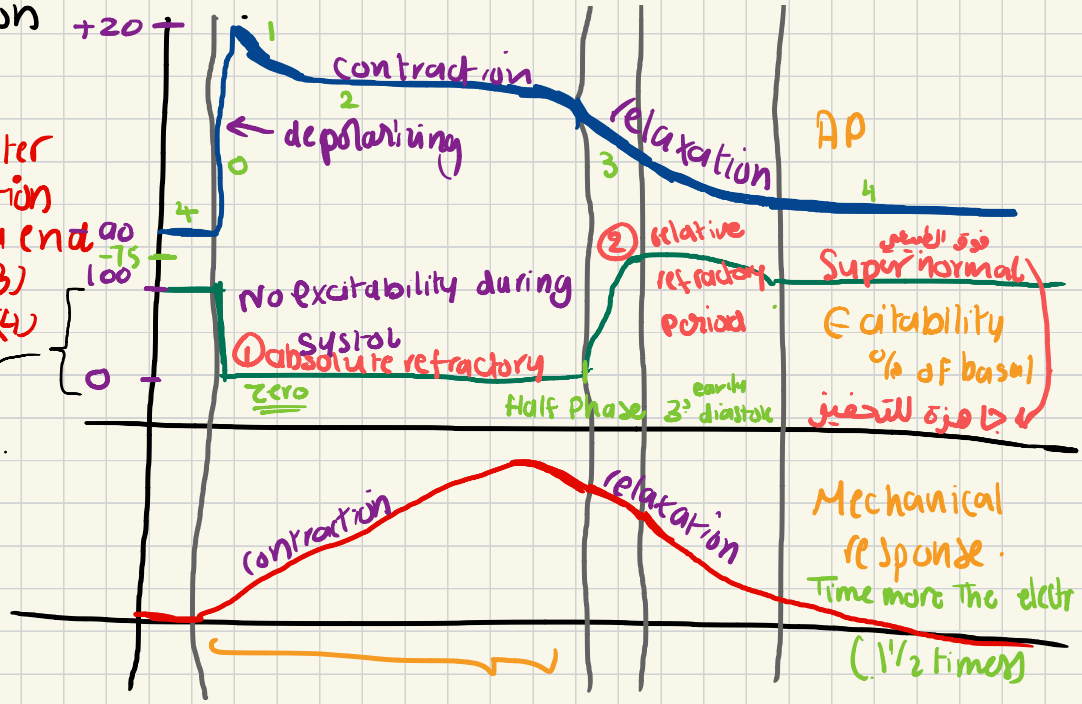
: Ability to respond to stimulation
- Cardiac response is by generating Exitatory-Conductive system \ fast AP response

→ fast response action potential
- Triphasic repolarization



Excitability & contraction relation.

→ Mechanical response: after 20 ms of depolarization
→ Systole (max) at plateau end
→ half diastole → phase (3)
→ half of diastole → phase (4)



★ Absolute refractory Period.
- Start of phase 0 → Phase 3 'middle'
- Excitability = Zero 0
- Prevent heart being tetanized.
- Prevent fatigue

★ Relative Refractory period
- excitability gradually restored
middle of Phase 3 → repolarize -75mv

★ Super normal Phase: → ؟ خوف قُ record.
- Excitability higher than normal
- during late phase 3.

& Ninja Nerd

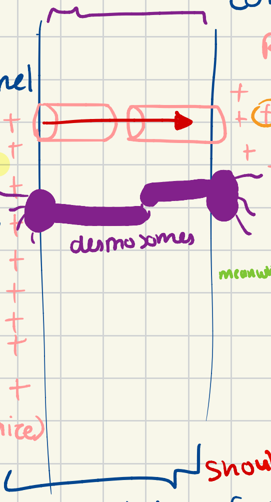
How action potential happening?

Nodule cell

I) funny Na^+ channel
Slow flow.
-60 mv to -55mv
II) T-type Ca^+ channel
stimulate to open
-55mv to -40mv
Threshold to open Ca^+ channels which Burst.
III) L-type Ca^+ channel
∴ +ive inside cell to +40 mv. (depolarize)

No RMP
But they have membrane potential {before Na^+ channels open}
L -60 mv.

Intercalated disk (desmo + Gap junction)



connected by Gap Junction
should be close together?
done by desmosomes

Contractile cells
Resting M Potential = -85 \ -90 mV
① → Bring to Threshold ?-70 mv
∴ Special voltd Na^+ channel open
V. fast flow! V. tive
meanwhile: Some Ca^+ open
↓
to +10 mv?

Q: rythmicity in SA node is 100\min
resting heart rate is 75 beat\min
why?
- due to continuous inhibitory discharg from vagus nerve. ∴ decrease 100 - 75
Called Vagal Tone. لانو ال Symp بعطي اكثر من
١٠٠ لو ضل الحاله ؛ و لازم نعوض vagus ← بتأثير ع نقص الحلقة.

Vagus supply the whole cardiac muscle except Ventricles
↓ {Vagal escape phenomenon}
protect from abnormal high vagal stimulation → cause Cardiac Arrest

Conductivity

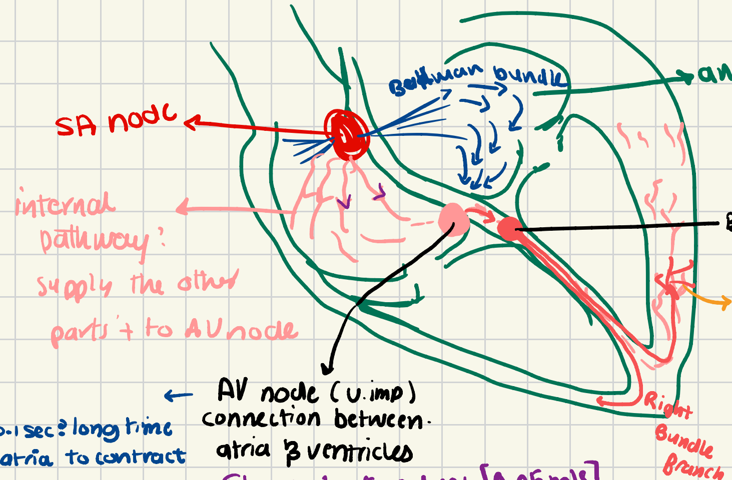
Cardiac muscle transmit waves through high specialized systems

Cardiac Conducting system {3 parts} Nodals – internodal – His · purkinji

SA \ AV

3 bundles:
1) ant.
2) middle
3) Post.
Consist (SA to AV)

1 – AV bundle
2 – R\L bundle branches
3 – Purkinje fibers



SA → AV : only 1 way conduction [→] only

internal pathway: supply the other parts + to AV node

Purkinji fibers: Convey excitation to ventricle's muscle.
Fastest conduction (4 m\s) {So both ventricles contract together}
* has ↑ Gap junctions * nature: very large fibers (↓ resistance)

Receive ← AV node (v.imp) connection between atria & ventricles

(why?) 1) Take 0.1 sec: long time
gives time atria to contract
↳ so push the blood

- few gap junction
- smaller diameter

– Slowest conduct [0.05 m\s]
– also named: electric isolator.

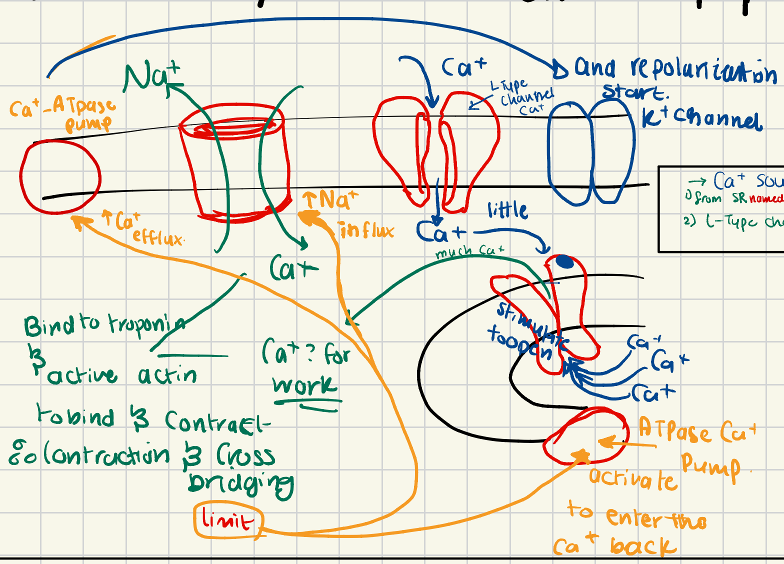
Significance:
1) → protect ventricles from pathological atrial rythm.
2) [Prevent ventricle fibrillation] ↳ يمر max impulse 230 impulse\min

Contractility

: ability to contract responding to stimulus

Change chemical energy → Mechanical work

Excitability \ contraction coupling



→ Ca+ sources ?
1) from SR named: activator Ca+
2) L-type channels named: depolarized Ca+

Factors effecting

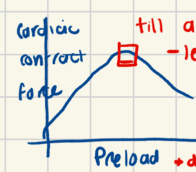
Mechanical

regarding the action

[1] Preload → EDV [2] after load

↑EDV = ↑ initial length fiber

↑ tension & ↑stretch
↑ contraction
(↑ velocity of shortening)



– length-tension relation
– Protational relation
– its the stroke volume
+ describe stroke & EDV relation

-initial length GIs diastolic filling

over strength : marked decrease in EDV

Significance: Heterometric autoregulation

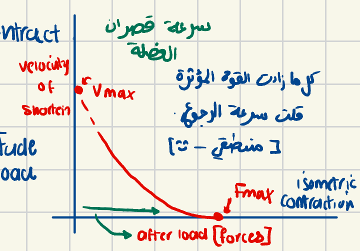
Cardiac

Extra-Cardiac

{in Table}

[2] After load

→ load when it begins to contract
→ Changes here affect the velocity of shortening of muscle :
initial velocity ∝ 1 / magnitude of afterload



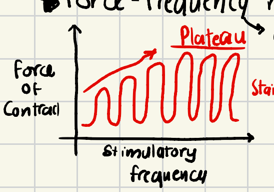
سرعة قصيران العضلة
كل ما زادت القوة المؤثرة
قلت سرعة الرجوع
[ــ – عكسي]

[2] Cardiac 'affect the cardiac performance'

(1) myo cardial mass Normal = max contraction force
(2) Heart rate
(3) Cardiac inotropic : state of cardiac loading state
حالة عضلة القلب

major determinants:
1) Ca+ quantity 2) myocardial mass
3) Extra cardiac factors.

force - frequency relationship



Stair case phenomen

↑ Ca+ influx till a level: No more Ca+
Tachycardia: +ive intropic
Bradycardia: -ive intropic

Extrinsic factors affecting

Factor	Rythmicity Chronotropism +	Rythmicity Chronotropism −	Excitability Bathmotropism +	Excitability Bathmotropism −	Conducting dromotropic +	Conducting dromotropic −	Contracting intropic +	Contracting intropic −
Nervous: para sy		✔		✔		✔		✔
Nervous: symp	✔	(How?) *	✔		✔		✔ ↑Ca+\L channel	
Chemical	Catecholamine Thyroxin Blood gases: mild-moderate hypoxia & hypercapnea	Ach blood gases: Severe O_2↓/ H^+↑/CO_2↑	Catecholamine Thyroxin	Hypoxia ischemia [Calcium: Hyper. can cause cardiac arrest	Catecholamine + Thyroxine Alkalosis	most electrolyte disturbances ((K+)) acidosis	-Catecholamine -Thyroxine -Glucagon -moderate O_2↓\ CO_2↑ - Hypercalcemia	-Excess Na^+ -Hyper Kalemia -Extreme O_2↓\ CO_2↑ -Acetyl choline ↳vagus
Drugs if	Symph mimetics	Digilatis (↓HR) Cholinergic	Xanthines	Cholinergic	Symp-mimetics	Cholinergic + Digitalis	Digitalis Xanthines (inhibit cAMP Break) (Caffeine \theoph)	-Calcium channel blockers inhibit L-channels-
Physical: ↑Body Temp	✔		✔		✔		✔	
Physical: ↓Body Temp		✔		✔		✔		✔

Sympathetic: epinephrine \ Nor → Bing to β1 receptor → activate intracellular med → ↑CAMP → activate PKA ((Target special protein channel)) → [↑Ca^+]

Digitalis
→ inhibit Na^+\ K^+ pump
→ ↑Na^+ inside
→ stimulate Na^+\ Ca^+ Exchang
→ ↑ Ca^+ intracellulary